

ation functional class using the PARTNER (Placement of Aortic Transcatheter Valves) data, but we would expect them to move in directionally similar ways, particularly for measures such as the physical component score of the SF-12 or SF-36. Longer term quality-of-life data from the PARTNER program are being collected but are not yet available for analysis. In the meantime, New York Heart Association data through 2 years have been reported for both the inoperable (2) and high-risk surgical candidate populations (3). These reports show that about 85% of 2-year survivors were in New York Heart Association functional class I or II after either transcatheter aortic valve replacement (TAVR) or aortic valve replacement, with no suggestion of deterioration from year 1 to year 2. However, these analyses of survivors may be affected by attrition of the sickest patients over time. Given the age and medical complexity of patients currently undergoing TAVR, it would not be surprising to see some decline in functional status and quality of life over time, as observed with aging in general, and as suggested in a UK analysis of TAVR patients limited to those who completed surveys at all 4 time points in the first year (4). We agree that the identification of baseline patient factors that reliably predict changes in health status after TAVR (or aortic valve replacement) would aid in refining patient selection for these interventions and should be the focus of subsequent investigations.

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REFERENCES

1. Reynolds MR, Magnuson EA, Wang K, et al., for the PARTNER Trial Investigators. Health-related quality of life after transcatheter or surgical aortic valve replacement in high-risk patients with severe aortic stenosis: results from the PARTNER (Placement of Aortic Transcatheter valve) trial (Cohort A). *J Am Coll Cardiol* 2012;60:548–58.
2. Makkar RR, Fontana GP, Jilaihawi H, et al. Transcatheter aortic-valve replacement for inoperable severe aortic stenosis. *N Engl J Med* 2012;366:1696–704.
3. Kodali SK, Williams MR, Smith CR, et al. Two-year outcomes after transcatheter or surgical aortic-valve replacement. *N Engl J Med* 2012;366:1686–95.
4. Fairbairn TA, Meads DM, Mather AN, et al. Serial change in health-related quality of life over 1 year after transcatheter aortic valve implantation: predictors of health outcomes. *J Am Coll Cardiol* 2012;59:1672–80.

Outcomes of Atrial Fibrillation Ablation in Patients With Metabolic Syndrome

We read with interest the paper by Mohanty et al. (1), which demonstrates a strong association between metabolic syndrome

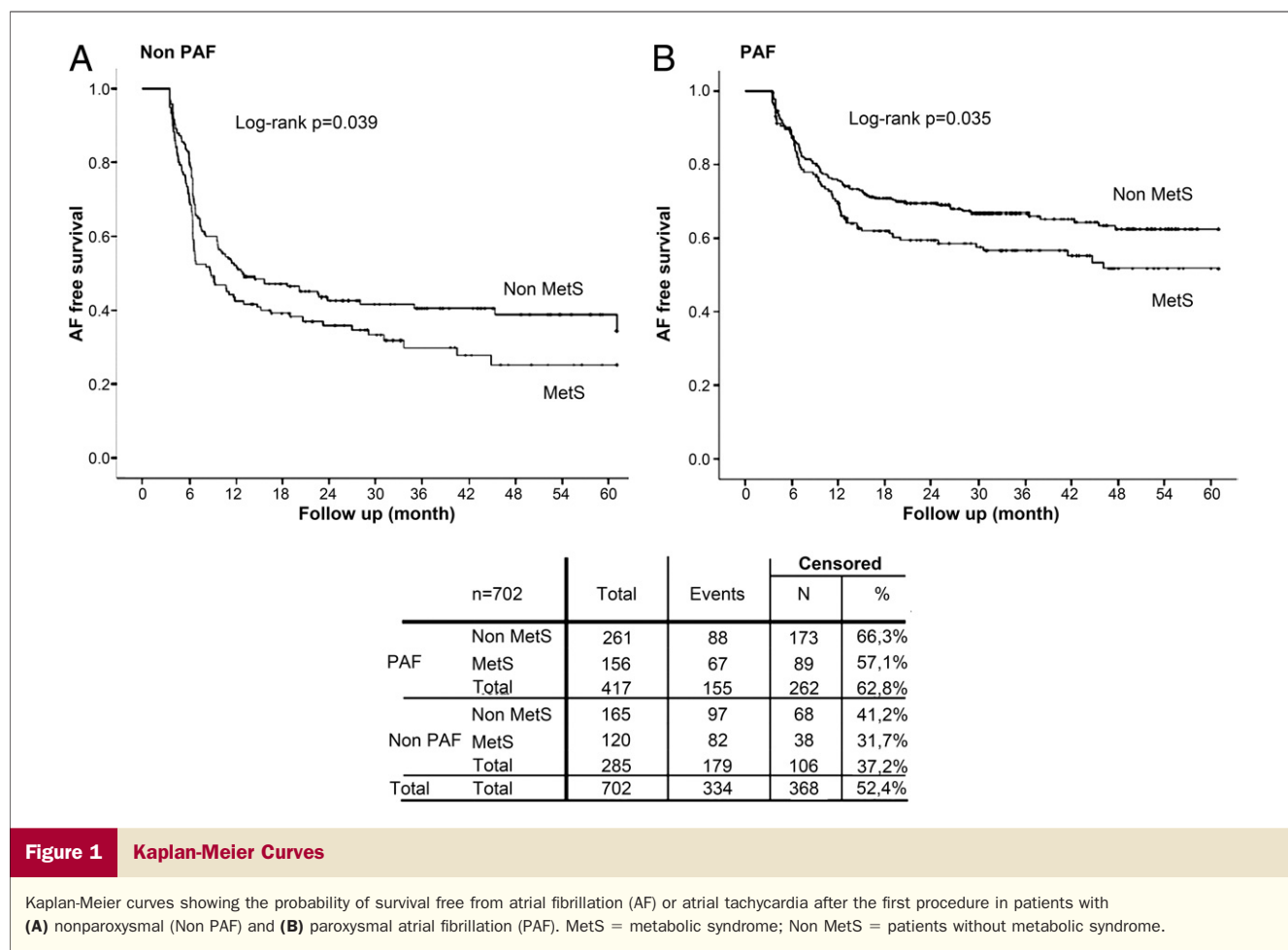
(MS) and recurrence of atrial fibrillation after catheter ablation (AFCA) but only in patients with nonparoxysmal atrial fibrillation (NPAF).

Recently, we presented the data of 5-year follow-up in a group of 702 patients after both radiofrequency and cryoballoon AFCA that proved MS to be the independent predictor of atrial fibrillation (AF)-free survival AFCA, regardless of left atrium size (LAS), type of AF, and energy source used (2). Patients with MS were 1.32× more likely to have AF recurrence than patients without MS. Median time to AF recurrence AFCA was 18.6 months in patients with MS and 28.6 months in patients without MS ($p = 0.011$) (Fig. 1). Similar results were published by others (3,4), and the discrepancy had already been mentioned by Asirvatham and Jiao (5). Interestingly, our data showed that MS had no impact on outcome after re-do pulmonary vein isolation, which might support the editorial comment: “perhaps we learned that trigger elimination, even if in 1-time procedure, can be effective despite an ongoing primary arrhythmia-provoking process” (5).

The contradictory findings by Mohanty et al. (1) might be (in part) explained by several limitations of their study. First, the presented study group was not as homogenous as ours, with significant differences in LAS and upstream therapy drugs. The LAS is known to be one of the strongest predictors of outcome after AF ablation (6). If LAS was significantly bigger in NPAF without MS, the expected results could have been as described by investigators (1). Renin-angiotensin-aldosterone system blockers protect from AFCA in patients with PAF (7). Most of the patients with MS (82%) received angiotensin-converting enzyme inhibitors compared with patients without MS (28%). If the similar proportion was in the PAF group, we could have expected better outcome in patients with MS (i.e., no significant difference as described) (1). The role of statins post-pulmonary vein isolation has not been established (6), but the published meta-analysis reported statins to be more effective for prevention of PAF (8). Again, a higher number of patients with MS (91%) received lipid-lowering therapy than patients without MS (30%). Second, the results should be checked—keeping in mind the noncompletely homogenous study group—with a propensity score matching, which allows for analysis in a well-balanced cohort. It would be interesting to know whether Mohanty et al. (1) would reach the same results in the well-matched samples. Third, the follow-up period was (only) 21 ± 7 months. The difference in the outcome AFCA in patients with and without MS becomes even more significant within longer follow-up, both in PAF and NPAF groups (2).

Following the latest recommendations (6), we think that it is important to recognize that AF recurrence rates AFCA depend on concomitant diseases, and outcome of AFCA in patient populations not well-represented in clinical trials should be reported. Patients with MS are such a population. Therefore, further discussion and clear data presentation are needed to solve the discrepancy in reported results.

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REFERENCES

- Mohanty S, Mohanty P, Di Biase L, et al. Impact of metabolic syndrome on procedural outcomes in patients with atrial fibrillation undergoing catheter ablation. *J Am Coll Cardiol* 2012;59:1295–301.
- Berkowitsch A, Kuniss M, Greiss H, et al. Impact of impaired renal function and metabolic syndrome on the recurrence of atrial fibrillation after catheter ablation: a long term follow-up. *Pacing Clin Electrophysiol* 2012;35:532–43.
- Tang RB, Dong JZ, Liu XP, et al. Metabolic syndrome and risk of recurrence of atrial fibrillation after catheter ablation. *Circ J* 2009;73:438–43.
- Chang SL, Tuan TC, Tai CT, et al. Comparison of outcome in catheter ablation of atrial fibrillation in patients with versus without the metabolic syndrome. *Am J Cardiol* 2009;103:67–72.
- Asirvatham SJ, Jiao Z. What causes atrial fibrillation and why do we fail with ablation? Insights from metabolic syndrome. *J Am Coll Cardiol* 2012;59:1302–3.
- Calkins H, Kuck KH, Cappato R, et al. 2012 HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation: recommendations for patient selection, procedural techniques, patient management and follow-up, definitions, endpoints, and research trial design. *J Interv Card Electrophysiol* 2012;33:171–257.

- Berkowitsch A, Neumann T, Kuniss M, et al. Therapy with renin-angiotensin system blockers after pulmonary vein isolation in patients with atrial fibrillation: who is a responder? *PACE* 2010;33:1101–11.
- Savelieva I, Kourliouros A, Camm J. Primary and secondary prevention of atrial fibrillation with statins and polyunsaturated fatty acids: review of evidence and clinical relevance. *Naunyn Schmiedebergs Arch Pharmacol* 2010;381:1–13.

Reply

We thank Dr. Wojcik and colleagues for their interest in our study (1).

The apparent contradiction between our findings and their results can be explained by at least 3 factors: difference in study population, detection and elimination of nonpulmonary vein (non-PV) triggers, and use of discretion of the physician in patient selection.

Our population had predominantly nonparoxysmal atrial fibrillation (NPAF), compared with theirs. Most importantly, we observed in the NPAF cohort significantly more frequent non-PV triggers in the metabolic syndrome (MS) group compared with the non-MS group, which is in accordance with earlier studies by others (2). As we discussed in our study, the challenge of eliminating non-PV triggers could have contributed to the higher recurrence in the MS group of the NPAF cohort. Of note,